Exhibit G

Acidosis and Catecholamine Evaluation Following Simulated Law Enforcement "Use of Force" Encounters

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Abstract

Objectives: Law enforcement authorities are often charged with controlling resisting suspects. These encounters sometimes result in the sudden and unexpected death of the suspect. Drug intoxication, excited delirium syndrome, or excessive uses of force are factors that are often blamed, but sometimes the mechanism of these deaths is not fully understood. It is possible that worsening acidosis or excessive catecholamine release play a part. The objective of this study was to determine the effect on markers of acidosis and catecholamines of various tasks intended to simulate common arrest-related situations.

Methods: Subjects were assigned to one of five task groups: 1) a 150-meter sprint and wall hurdle (simulated flight from arrest); 2) 45 seconds of striking a heavy bag (simulated physical resistance); 3) a 10-second TASER X26 electronic control device exposure; 4) a fleeing and resistance exercise involving a law enforcement dog (K-9); or 5) an oleoresin capsicum (OC) exposure to the face and neck. Baseline serum pH, lactate, potassium, troponin I, catecholamines, and creatine kinase (CK) were evaluated. Serum catecholamines, pH, lactate, and potassium were sampled immediately after the task and every 2 minutes for 10 minutes posttask. Vital signs were repeated immediately after the task. Serum CK and troponin I were evaluated again at 24 hours posttask.

Results: Sixty-six subjects were enrolled; four did not complete their assigned task. One subject lost the intravenous (IV) access after completing the task and did not have data collected, and one subject only received a 5-second TASER device exposure and was excluded from the study, leaving 12 subjects in each task group. The greatest changes in acidosis markers occurred in the sprint and heavy bag groups. Catecholamines increased the most in the heavy bag group and the sprint group and increased to a lesser degree in the TASER, OC, and K-9 groups. Only the sprint group showed an increase in CK at 24 hours. There were no elevations in troponin I in any group, nor any clinically important changes in potassium.

Conclusions: The simulations of physical resistance and fleeing on foot led to the greatest changes in markers of acidosis and catecholamines. These changes may be contributing or causal mechanisms in sudden custodial arrest-related deaths (ARDs). This initial work may have implications in guiding applications of force for law enforcement authorities (LEAs) when apprehending resisting subjects.

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Sudden and unexpected deaths have occurred in custodial situations for centuries.[1] The mechanisms of many of these deaths remain unclear. These types of deaths in modern-day society are most often associated with violent and resistive encounters with law enforcement authorities (LEAs) attempting a custodial arrest. These arrest-related deaths (ARDs) tend to yield scrutiny of the LEAs' tools and tactics used in the encounter.

There has been speculation about the mechanisms responsible for ARD. While drug intoxication, excited delirium syndrome, or LEA excessive force practices have been implicated, the possible mechanisms leading to ARD have not been well studied. Worsening acidosis and a hyperadrenergic state are two possible mechanisms.[2,3] In this study, we examined these parameters within the context of commonly used tools, tactics, and suspect behaviors to establish a better understanding of the physiologic state of a person at the time of ARD. The objective of this study was to determine the effect on markers of acidosis and catecholamines of various tasks intended to simulate common arrest-related situations.

METHODS

Study Design

This was a prospective, experimental study of human subjects. All subjects provided informed consent. The institutional review board at Hennepin County Medical Center approved the study.

Study Setting and Population

The study was conducted at the Thomas A. Hontz Police and Fire Training Facility (Scottsdale, AZ) in the context of a LEA training environment. Data gathering took place in March 2009 during daylight hours. Daily outdoor temperatures ranged from 43.3 to 76.5° F over the 3 days of testing.

The participants were a convenience sample of law enforcement and corrections officers, non-LEA public safety personnel, TASER International, Inc., employees, and academic researchers participating in a training event sponsored by TASER International, Inc. (Scottsdale, AZ). Attendees at the training event were notified of the research and instructed to contact the investigators if interested. All participants were aware that they were participating in training that could subject them to various uses of force, including a TASER device exposure. All of the training conditions used in this research are common situations used in the training of police and corrections officers and were familiar to persons at this event. This training event was chosen as a recruitment site because participants had familiarity with the events in each arm of the study, allowing them to make informed decisions about enrollment in the study.

Each subject was asked to complete one of five tasks meant to simulate various custodial arrest-related conditions. Each subject then completed a medical screening questionnaire describing medical history, current medications, and recent exercise, which was reviewed by a study physician. Inclusion criteria were the ability to participate in vigorous physical exercise. Known pregnancy was the only exclusionary criteria. Each subject was given a TASER device as compensation for participation at the completion of the study.

Study Protocol

Each subject was prospectively assigned to one of five tasks that were meant to simulate suspect behaviors that commonly occur or the LEA use of tools and tactics that are commonly used during custodial arrest situations of a resisting subject: 1) 150-meter sprint and wall hurdle, simulating flight on foot from LEAs; 2) 45 seconds of hitting and kicking a heavy bag, simulating the physical exertion of resisting arrest by LEAs; 3) 10-second continuous TASER X26 device exposure; 4) an LEA-trained dog (K-9) resistance exercise simulating fleeing on foot from and resisting a K-9; or 5) an oleoresin capsicum (OC) foam exposure to the face/neck.

Subjects assigned to the K-9 resistance task group were screened first for prior K-9 training or handling experience. Subjects who had prior K-9 training or handling experience were not eligible for the K-9 resistance task and were assigned the next available task. All subjects were asked not to engage in any physical exercise regimens for 48 hours prior to testing and until their final blood draw 24 hours after the task.

Details of the Task Groups

Task Group 1 (150-meter Sprint and Wall Hurdle). The subject sprinted 150 meters in a straight line on a blacktop covered level surface. The subject had 10 yards at the end of the sprint to slow and prepare for a wall hurdle. The wall hurdle required the subject to jump/climb over a 44-inch wall. The time to complete the task was recorded. The subject was encouraged verbally during the event.

Task Group 2 (45 Seconds of Heavy Bag Physical Resistance). The subject was required to keep a suspended heavy bag away from him- or herself by any means possible. This included punching, kicking, head butting, and throwing knees and elbows offensively at the bag. The subject had the option to use any or all of these methods. The subject wore athletic shoes, had hands wrapped with protective athletic tape during the task, and was encouraged verbally during the event.

Task Group 3 (10-second TASER X26 Device Exposure). The subject was exposed to a 10-second continuous TASER X26 electronic control device discharge to the back with deployed probes. The probes were deployed with the subject in the prone position on a protective mat from a distance of approximately 7 feet; the operator fired from an elevated position on a step-ladder. The probes and the TASER device were standard products from the manufacturer. The spread between the probes was measured to indicate the area of the subject exposed to the current.

Task Group 4 (K-9 Fleeing and Physical Resistance Exercise). The subject wore a protective suit. The two K-9s used in this task were both on active police duty.

Their handlers were with them during the entire task. The subject sprinted 160 feet in front of and away from the K-9. Near the completion of the sprint, the K-9 was released to chase the subject. The subject was instructed to present an arm for the K-9 to bite when nearing the completion of the sprint. The K-9 stayed on the bite for 20 seconds and the subject had been instructed to attempt to resist the K-9 during this time by pulling away and trying to stay on his or her feet. The K-9 handler provided verbal commands to the K-9 and the subject, simulating an arrest situation. The total task time was approximately 30 seconds.

Task Group 5 (OC "Pepper Spray"). The subject was sprayed with 10% OC foam (Sabre Red, Security Equipment Corp, Fenton, MO) for a period long enough in duration to cover the face and the anterior portion of the neck (approximately 2–3 seconds). The subject was sprayed with eyes and mouth closed and was allowed to rinse the foam off with water after 10 seconds of exposure time. The subject was allowed access to continuous fresh air, running water, and a cooling fan if desired following the exposure.

Measures

Each participant had an 18- or 20-gauge intravenous (IV) catheter placed in the left or right antecubital fossa by a physician or paramedic prior to the test. Baseline automated blood pressure and heart rate were recorded by a Nonin 2120 device (Nonin Medical, Inc., Plymouth, MN). Baseline venous blood specimens for measurement of serum catecholamines (epinephrine, norepinephrine, dopamine, and total), pH, lactate, potassium, troponin I, and creatine kinase (CK) were obtained through the IV catheter.

Each subject then participated in his or her assigned task. Catecholamines, pH, lactate, and potassium were drawn immediately (within 30 seconds) after the task and every 2 minutes until 10 minutes posttask. Vital signs were repeated immediately (within 30 seconds to 1 minute) after the task. CK and troponin I were obtained again at 24 hours. All samples for fractionated catecholamines, potassium, troponin, and CK were centrifuged and held on ice for off-site analysis and determination (LabCorp, Inc., Phoenix, AZ). Venous pH and lactate were determined on-site, using the i-STAT system and CG4+ cartridges (Abbott Point-of-Care, East Windsor, NJ).

Data Analysis

On-site data were compiled in an Excel spreadsheet (Microsoft Corporation, Redmond, WA). Data were exported into STATA 10.0 (StataCorp, College Station, TX) for analysis and SigmaPlot 11 (Systat Software, Inc., San Jose, CA) for graphical presentation. Descriptive statistics were used where appropriate. Data were not normally distributed and are reported as medians and ranges. Proportions describing subject characteristics were compared using chi-square tests. Values at each time point were compared between groups using the Kruskal-Wallis equality of populations rank test. Within each group, the baseline value was compared to subsequent values using Wilcoxon signed-rank tests. Catecholamine values were reported as means with standard errors of the mean. To detect a 5% difference in the pH value between the groups at any time point, assuming a standard deviation (±SD) of 5%, with a significance of 0.05, and a power of 90%, 11 subjects were needed in each group.

RESULTS

Sixty-six subjects were enrolled. Three subjects were disqualified secondary to an inability to obtain IV access, and one of them had vasovagal syncope associated with multiple attempts to place an IV catheter. One subject was excluded secondary to refusal to participate in the prospectively assigned group. This subject had significant anxiety leading to vasovagal syncope when told that he or she was being assigned to the OC foam exposure-task group.

Sixty-two subjects completed the assigned tasks. Two of these subjects had their data excluded: one subject assigned to the heavy bag group had an IV catheter failure after the task, and further attempts at catheter placement were unsuccessful. The other subject was in the TASER X26 exposure group and had a 5-second continuous exposure instead of a 10-second exposure due to operator error. Data from both of these subjects were excluded from the group analysis (Table 1).

There were 12 subjects in each of the task groups for the final analysis. The subject characteristics and vital signs are presented in Table 2. There was no difference between the groups in age ($p = 0.31$), sex ($p = 0.10$), or body mass index (BMI; $p = 0.10$). Health histories included anxiety (n = 1), hypertension (n = 5), high cholesterol (n = 3), asthma (n = 1), chronic back pain (n = 1), gastroesophageal reflux disease (n = 1), depression (n = 3), Hashimoto's disease (n = 1), and hypothyroidism (n = 1). One subject had prior sex-reconstructive surgery, one had recent hand surgery, and one listed "low blood pressure" as a current medical condition. The median sprint task group time was 25.6 seconds (range 22.0–31.6 seconds). The median TASER X26 probe spread was 12 inches (range 6–13 inches).

One subject reported musculoskeletal shoulder pain after the TASER X26 device exposure that persisted, but was improved at 24 hours. On later telephone follow-up with this subject, there were no further complaints, and the issue had resolved. One subject in the TASER X26 device exposure task group had an apparent vasovagal syncopal episode after the completion of the exposure, but became responsive within 5 seconds with gentle stimulation and had no complaints upon recovery. Five of the heavy bag subjects had abrasions to the knuckles after the task. In addition, five of these subjects became nearly syncopal and vomited after the task and had to be placed supine to recover. One sprint group subject had a syncopal episode 8 minutes after his task (he had been lightheaded and nauseated since completing the task). No other adverse events were reported. All subjects recovered and felt as at baseline prior to releasing them from the testing site.

The pH and lactate values are presented in Tables 3 and 4. The groups were not different at baseline.

Table 1
Results From Excluded Subjects

Subject; Reason	Pre	Post	2 Minutes	4 Minutes	6 Minutes	8 Minutes	10 Minutes	24 Hours
Heavy bag #8; IV catheter failure	a) 299	a) 1,359	a) X	a) X	a) X	a) X	a) X	a) X
	b) 7.32	b) 7.11	b) X	b) X	b) X	b) X	b) X	b) X
	c) 2.04	c) 16.96	c) X	c) X	c) X	c) X	c) X	c) X
	d) 580	d) X	d) X	d) X	d) X	d) X	d) X	d) X
	e) 4	e) 3.8	e) X	e) X	e) X	e) X	e) X	e) X
	f) <0.2	f) X	f) X	f) X	f) X	f) X	f) X	f) X
TASER #1; 5-second exposure only	a) 590	a) 727	a) 697	a) 616	a) 621	a) 597	a) 630	a) X
	b) 7.367	b) 7.334	b) 7.331	b) 7.340	b) 7.335	b) 7.387	b) 7.387	b) X
	c) 1.65	c) 1.33	c) 1.46	c) 1.56	c) 1.73	c) 1.69	c) 1.69	c) X
	d) 106	d) X	d) X	d) X	d) X	d) X	d) X	d) 129
	e) 3.9	e) 3.4	e) 3.8	e) 3.7	e) 3.7	e) 3.9	e) 3.9	e) X
	f) <0.2	f) X	f) X	f) X	f) X	f) X	f) X	f) <0.2

a) Total catecholamines (pg/mL); b) pH; c) lactate (mmol/L); d) CK (U/L); e) K (mmol/L); f) troponin I (ng/mL); X = not obtained.

Table 2
Subject Characteristics and Vital Signs

	Group				
	Sprint	OC	TASER	Heavy Bag	K-9
Age, yr	36.5 (24–49)	40 (21–48)	34 (24–67)	36 (24–50)	26 (19–47)
Male sex, %	91.7	75.0	92.3	100	66.7
Median BMI	26.2 (20.9–31.9)	30.0 (26.2–39.3)	26.5 (22.1–36.9)	28.2 (21.7–44.1)	25.8 (19.1–42.8)
Pretest BP, mm Hg	128/73 (119/61–152/100)	148/98 (121/74–184/123)	139/88 (126/71–175/101)	141/92 (118/73–191/117)	129/85 (105/67–171/100)
Posttest BP, mm Hg	151/82 (87/65–198/100)	170/90 (106/85–202/130)	141/84 (122/87–178/86)	168/71 (108/95–202/111)	166/90 (133/92–192/100)
Comparison to baseline	0.011	0.013	0.564	0.035	0.004
Pretest HR, beats/min	72 (62–98)	91 (69–110)	92 (60–109)	74 (64–117)	80 (60–100)
Posttest HR, beats/min	130 (88–170)	88 (53–110)	96 (66–115)	146 (115–181)	122 (71–141)
Comparison to baseline	0.002	0.266	0.889	0.002	0.002

Values reported are median (range) unless otherwise noted.
BP = blood pressure; BMI = body mass index; HR = heart rate; OC= oleoresin capsicum.

Table 3
Median pH (Range)

Group	Baseline	Immediate Post	2-minute Post	4-minute Post	6-minute Post	8-minute Post	10-minute Post
Sprint	7.32 (7.30–7.44)	7.16 (7.05–7.31)	7.17 (7.09–7.31)	7.18 (7.11–7.28)	7.19 (7.12–7.29)	7.21 (7.13–7.31)	7.22 (7.16–7.32)
OC	7.36 (7.33–7.39)	7.37 (7.33–7.40)	7.37 (7.32–7.47)	7.39 (7.36–7.45)	7.37 (7.34–7.48)	7.36 (7.34–7.48)	7.37 (7.32–7.48)
TASER	7.37 (7.32–7.43)	7.29 (7.24–7.35)	7.29 (7.25–7.33)	7.32 (7.28–7.35)	7.33 (7.31–7.36)	7.34 (7.31–7.39)	7.36 (7.34–7.39)
Heavy bag	7.36 (7.28–7.39)	7.04 (6.95–7.18)	7.01 (6.91–7.09)	7.01 (6.91–7.09)	7.06 (6.94–7.11)	7.05 (6.96–7.13)	7.06 (6.99–7.15)
K-9	7.34 (7.30–7.40)	7.26 (7.14–7.36)	7.25 (7.17–7.35)	7.26 (7.17–7.34)	7.27 (7.18–7.35)	7.27 (7.20–7.35)	7.31 (7.22–7.38)
p-value*	0.07	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

OC = oleoresin capsicum.
*Kruskal-Wallis test.

The pH and lactate (normal pH values 7.35 to 7.45 and lactate 1–2 mmol/L) changed from baseline at every time point for all groups ($p < 0.001$ for each, Wilcoxon signed-rank test). There were no differences in potassium (normal 3.5 to 5.0 meq/L) between the groups at baseline (median 4.0 meq/L, range 3.6–4.6 meq/L; $p = 0.755$). The sprint group and TASER group had a decrease in potassium at all time points ($p < 0.01$). The maximum decrease from baseline for the sprint group was 0.2 meq/L (from 3.9 to 3.7 meq/L), and for the TASER group was 0.3 meq/L (from 4.0 to 3.7). The heavy bag group had a decreased potassium at the first four time points ($p < 0.01$; maximum decrease from baseline 0.4 meq/L, from 4.0 to 3.6 meq/L), no difference from

Table 4
Median Lactate, mmol/L (Range)

Group	Baseline	Immediate Post	2-minute Post	4-minute Post	6-minute Post	8-minute Post	10-minute Post
Sprint	1.19 (0.67–3.55)	10.98 (3.25–14.60)	12.74 (6.17–16.72)	13.26 (6.50–16.47)	13.93 (8.49–16.66)	13.02 (8.55–16.56)	11.47 (7.61–15.77)
OC	1.01 (0.76–2.03)	1.39 (0.6–2.39)	1.45 (0.61–2.40)	1.50 (1.07–2.15)	1.49 (0.65–2.13)	1.34 (0.65–2.10)	1.50 (0.72–2.67)
TASER	1.30 (0.81–1.93)	5.49 (1.33–7.18)	5.52 (1.46–6.66)	5.31 (1.56–6.16)	4.76 (1.73–5.67)	4.60 (1.69–5.29)	4.06 (1.69–4.78)
Heavy bag	1.44 (0.73–2.61)	15.46 (8.85–18.65)	17.22 (15.14–20.0)	17.88 (15–01-20.0)	17.09 (13.81–20.0)	18.26 (13.67–20.0)	17.33 (13.88–20.0)
K-9	1.05 (0.61–1.45)	5.01 (1.54–9.58)	5.76 (2.30–10.16)	6.13 (2.87–11.0)	6.53 (2.81–10.55)	6.34 (2.85–10.41)	5.90 (2.42–9.87)
p-value *	0.066	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

OC = oleoresin capsicum.
*Kruskal-Wallis test.

Table 5
Creatinine Kinase, U/L

Group	Baseline	24-hour Post	Difference From Baseline (Range)	p-value*
Sprint	144.8 (±55.4) 57–231	196.8 (±91.6) 57–440	30.5 (–19 to 205)	0.006
OC	215.3 (±145.8) 98–597	214.1 (±139.9) 69–508	5.5 (–120 to 92)	1.000
TASER	184.4 (±155.3) 42–576	241.1 (±133.5) 59–447	26 (–205 to 329)	0.25
Heavy bag	303.8 (±221.8) 99–758	351.6 (±285.3) 109–1161	74 (–160 to 403)	0.31
K-9	137.4 (±137.4) 58–366	174.8 (±170.5) 57–536	–3 (–32 to 322)	0.57
p-value†	0.038	0.157	0.70	

Values are reported as mean (±SD) range unless otherwise noted.
OC = oleoresin capsicum.
*Wilcoxon signed-rank test.
†Kruskal-Wallis test.

baseline at the 8-minute time point ($p = 0.475$), and an increase of 0.3 meq/L at the 10-minute time point (from 4.0 to 4.3 meq/L, $p = 0.002$). There were no "positive" troponin I values (normal < 0.4 ng/mL). CK data are presented in Table 5. No subjects had rhabdomyolysis based on a CK value greater than 5,000 U/L (normal 60 to 400 U/L). The highest CK value was 1,161 U/L in a heavy bag subject (his baseline value was elevated prior to the task at 758 U/L).

Catecholamine data are presented in Figure 1. Due to dilution-related difficulties, fully quantified values for catecholamines were not available for several subjects with very high levels. These were only reported as "greater than" the last value obtained before the quantity was not sufficient for further dilution. This limitation affected the sprint task group primarily ($n = 6$), but also affected the K-9 resistance task group ($n = 5$) and the heavy bag task group ($n = 4$). It did not affect the TASER group or OC group. For total catecholamines, there was no difference between the groups at baseline ($p = 0.769$). The values increased from baseline in all groups at all time points ($p < 0.002$ for all groups by Wilcoxon signed-rank test). For norepinephrine, there was no difference among the groups at baseline. The values increased from baseline in all groups at all time points ($p < 0.002$ for all groups by Wilcoxon signed-rank test). For epinephrine, there was a difference at baseline in epinephrine, which was noted to be highest in the TASER device exposure and the heavy bag groups. All groups had increases from baseline at time point 2 ($p < 0.001$); by time point 3 the TASER group was no longer increased from baseline ($p = 0.21$), but the other groups were ($p < 0.01$). By time point 4, the K-9 resistance ($p = 0.67$) and TASER device ($p = 0.78$) exposure groups were no longer increased from baseline, while the other groups were ($p < 0.01$); by time point 5 the OC exposure ($p = 0.31$), TASER device exposure ($p = 0.35$), and K-9 resistance ($p = 0.72$) groups were not changed from baseline. These groups remained unchanged from baseline at subsequent time points. The sprint and heavy bag groups remained increased from baseline at all time points ($p < 0.001$). For dopamine, there was no difference among the groups at baseline but there was a difference at all groups at each subsequent time point ($p < 0.02$).

DISCUSSION

Metabolic acidosis can cause autonomic instability, depressed myocardial function, arrhythmias, and cardiovascular collapse. There is literature to support metabolic acidosis as a possible mechanism of ARDs. Hick et al.[4] reported a series of five patients who sustained cardiac arrest temporally associated with LEA custodial arrest. The subjects had a pH range of 6.25 to 6.81. Four of the subjects died despite aggressive management. Gass et al.[5] found that lactate peaked at the sixth minute of inactive recovery in subjects completing a maximum exercise regimen on a motor-driven treadmill. The mean peak lactate was 14.2 mmol/L. Allsop

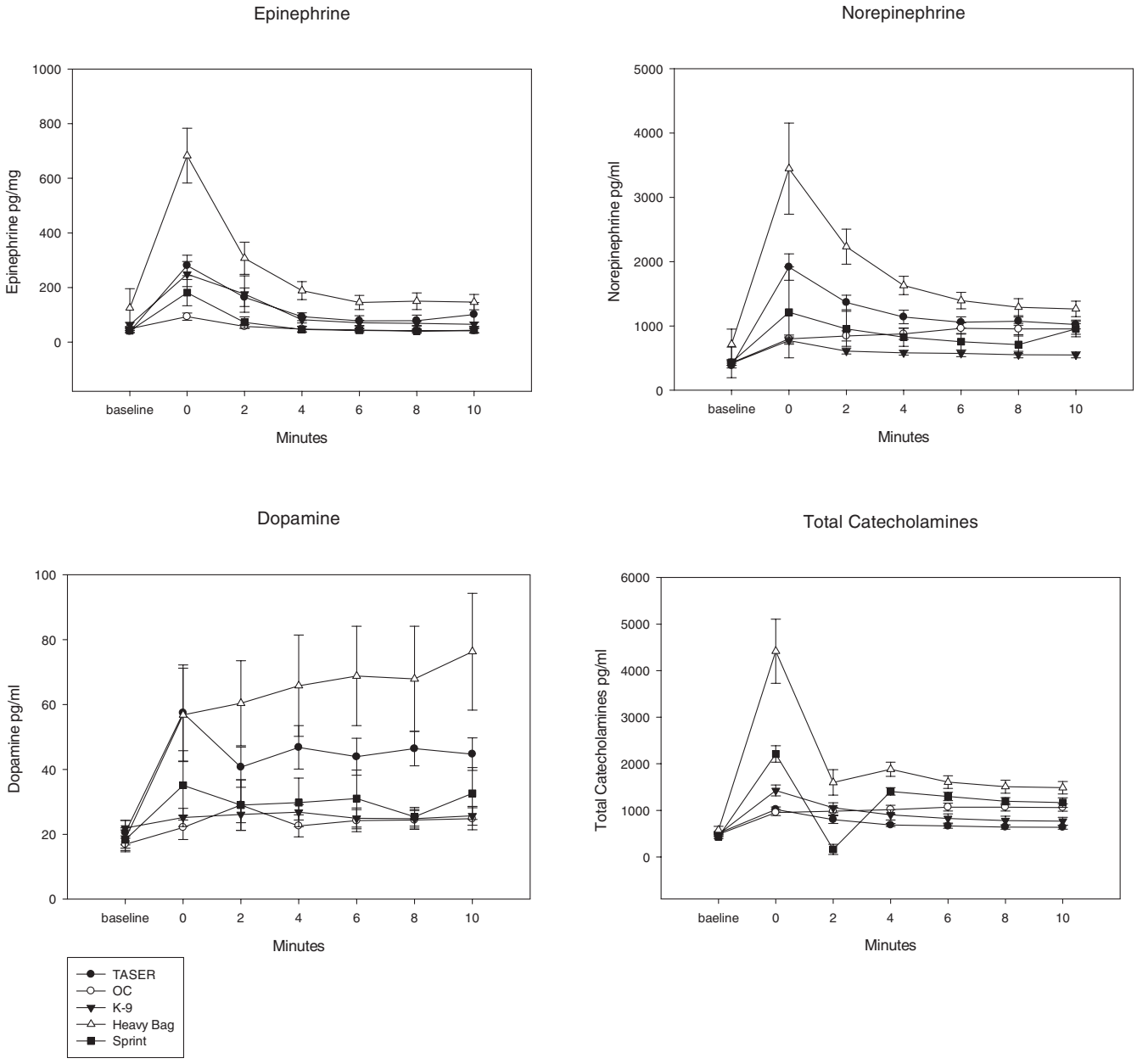

Figure 1. Catecholamines (pg/mL) mean value (error bar = SEM). OC = oleoresin capsicum.

et al.[6] found that venous pH decreased from 7.39 to 7.04 after a 30-second maximal sprint. The pH was 7.29 at 30 minutes. Lactate peaked at 15.76 mmol/L 5 minutes after the completion of the sprint, declining to 10.30 mmol/L at 30 minutes.[6] In our study, we found decreases in pH after tasks designed to simulate common subject behaviors and tools and tactics used on resistive subjects by LEAs during a custodial arrest situation. The pH was lowest and the lactate was highest in the heavy bag resistance task group, followed by the sprint group.

The phenomenon of ARD is often associated with behaviors that can contribute to inducing or worsening acidosis. These factors include illicit stimulant abuse, agitated behavior, excited delirium syndrome, and heavy physical resistance to LEA or health care personnel attempting to control a person in this condition to render aid.[2,3,7] Our study demonstrates that physical resistance and fleeing may be significant contributors to acidosis. Based on this, the intuitively dangerous concepts of running from and resisting LEAs or LEA K-9s appear to be true at a metabolic level. The important implications of this are that of the actions studied, the top three that appear to worsen acidosis are under the control of the subject and not the LEAs.

We believe that our data support the concept that LEAs should attempt to limit the length of time that a suspect is allowed to vigorously or violently resist restraint, and that once restrained, continued resistance should be viewed as potentially harmful and may require emergent medical intervention. Our study indicates that continued vigorous exertion that may be

typically displayed by an agitated, intoxicated, and delirious suspect may worsen acidosis and play a role in ARD.

There is also literature supporting a direct correlation between worsening acidosis and increasing catecholamine levels. A study by Goldsmith et al.[8] showed that for a given level of exercise, acidosis significantly correlates with an increasing rise of systemic catecholamines. Dimsdale et al.[9] noted increases in catecholamines in the period immediately after the cessation of exercise and hypothesized this as a mechanism for the increased risk of cardiac arrhythmias and ischemia in the period immediately after strenuous exercise. This hypothesis may be a factor in the sudden and unexpected nature of ARDs, which often involves cardiovascular collapse in the period just after the subject is restrained following significant physical exertion such as resistance and fleeing.[10,11] Some authors have hypothesized that this is related to a hypersympathetic state and an acute stress cardiomyopathy.[12–14] The phenomenon of acute stress cardiomyopathy has been referred to as ''tako-tsubo'' cardiomyopathy in Japan and has also been reported in the United States.[15] However, acute stress cardiomyopathy is not regularly described as progressing to sudden death.

Another theory linking catecholamines and ARDs suggests that the postexercise period might pose a risk because of the peripheral arteriolar dilation associated with exercise.[16] The vasodilation, coupled with a sudden decrease in venous return from the termination of muscular activity, may reduce cardiac output suddenly and reduce coronary artery perfusion at a time when the heart rate and oxygen demand are elevated due to high catecholamine levels.[17,18]

A prior study by Dawes et al.[19] used salivary amylase and cortisol as measures of the sympathetic-adrenal-medulla (SAM) axis and hypothalamus-pituitary-adrenal (HPA) axis responses to a prolonged TASER X26 device exposure, OC exposure, a defensive tactics drill, and the cold pressor task. This study demonstrated that OC exposure and physical exertion activated the SAM and HPA axes, similar to our findings. Increases in plasma catecholamines following physical exertion have been found previously by Allsop et al.[6] as well, who found larger changes than those detected in our study.

According to Karch,[20] under ''normal exercise conditions'' (not described as maximal), epinephrine levels are 700 pg/mL, and norepinephrine 299–300 pg/mL. In heart failure, they are 35–75 and 500–800 pg/mL; in cocaine use, 30–40 pg/mL and 1000–2000 pg/mL; and in cardiac arrest, 10,000–100,000 pg/mL and 500–600 pg/mL. However, Karch's data are based on only a single-time-point analysis. There is also evidence of elevations in catecholamines with animal-based restraint stress models (300%–600% increase) and with exercise stress combined with cocaine exposure (200%-500% increase), similar to the heavy bag group (600% increase in total catecholamines after the task) found here.[21,22]

It is possible that catecholamine excess, when combined with exertional stress during restraint, may decrease electrical cardiac stability making the myocardium more sensitive to arrhythmogenic drugs.[23] This may have important implications, because the majority of subjects in ARDs test positive for stimulant drugs in their system at time of autopsy.[11,24–26] However, Lakkireddy et al.[27] showed a 50% to 100% *increase* in the ventricular fibrillation threshold to a TASER device electrical shock in the setting of cocaine intoxication in a swine model.

We evaluated CK because rhabdomyolysis is a common complication of heavy exertion.[28,29] Heavy exertion from agitation, delirium, and custodial resistance is often described as a feature associated with ARDs. It has also been theorized that because a TASER works by causing skeletal muscle activation it might also cause significant rhabomyolysis. In a 2006 study by Ho et al.,[30] the mean change in CK at 24 hours was 57.2 U/L with a 5-second TASER X26 device-deployed probe exposure to the back. In our current study, the highest value was 1,161 U/L in a heavy bag subject. The highest median change from baseline in CK occurred with the heavy bag group followed by the sprint group, but only the sprint group change was significant. Our data suggest that rhabdomyolysis that is present in association with a resistive LEA interaction is likely due to volitional fleeing and resistance rather than LEA tools and tactics.

Hypokalemia in the period immediately after the cessation of exercise has been proposed as a possible contributory factor in ARDs. This potential ''period of peril'' has been thought to occur when exertion ceases because catecholamine-induced potassium absorption by cells continues.[2] The potassium changes we found, however, were not clinically significant. Our data do not suggest that hypokalemia plays a role in the mechanism of ARDs.

Our results for the TASER device exposure group were consistent with prior studies that showed minimal serum pH, lactate, and potassium changes and no associated troponin I elevations.[30–32] Our pH and lactate changes were more pronounced than those that Vilke et al.[31] found for the TASER, but we sampled pH values almost immediately after the exposure and used a longer exposure time. Prior research has shown that in exhausted subjects, TASER exposures are not associated with worsening acidosis differently than continued exertion.[33,34]

Oleoresin capsicum exposure appeared to cause continued rising catecholamine levels. This may be important because this effect could continue long after the subject was restrained. In addition, OC exerts its primary effect by pain and disorientation (due to an inability to open the eyes) and can also cause brief laryngospasm and bronchospasm.[35] This effect may cause even more release of catecholamines in a subject in a noncontrolled setting, especially in a subject with agitated delirium, already exhibiting paranoid behavior. Finally, it is theoretically likely that OC application in a field situation could result in a subject fleeing due to pain, further exposing him or her to the physiologic responses we saw in our Task Group 1 (sprint).

In ARD studies, death has usually been preceded by significant physical exertion and restraint.[4,10,11,13] Our data suggest that the most deleterious factor in LEA/subject encounters is physical resistance (heavy bag group) followed by fleeing on foot (sprint group).

These simulations resulted in the highest catecholamine levels and the greatest change in pH, lactate, and CK. In studies that have reviewed initial cardiac rhythms following collapse in ARD situations, the vast majority are pulseless electrical activity or asystole.[4,11,36] These terminal rhythms are consistent with severe acidosis that could occur in a prolonged or intense struggle.

LEAs are often challenged with protecting the public from agitated, violent subjects. The results of our study may be important in considering the best methods to ensure safety in custodial arrest situations and prevention of ARDs. We believe that our study demonstrates subject physiology during these encounters. These encounters cannot be without risk. When LEAs are called to deal with resistive subjects, the risk for injury and death to both the subject and the LEAs is elevated. The TASER device, K-9, and OC groups show that LEA intervention with these tactics and tools may have less negative consequences for acidosis and catecholamine levels than physical resistance or allowing the subject to flee and may be safer approaches to restraint.

LIMITATIONS

The volunteers were assigned to task groups based on testing station availability, and volunteers with prior significant LEA K-9 experience did not participate in the K-9 resistance task, introducing selection bias. Also, the number of volunteers was not large, limiting our ability to detect differences between groups. The number of volunteers in the study was kept to a minimum due to the logistical difficulties in carrying out the five tasks. Although the numbers are small, we believe that our data show consistent and important differences between groups. We encourage further investigation in this area.

Many of the subjects with very high catecholamine levels did not have fully quantified results. This affected the sprint, K-9 resistance, and heavy bag task groups, but not the TASER device exposure or OC exposure task groups. Because a true maximum quantification could not be performed for these three groups, we have underestimated the catecholamine levels in these three groups and limited our ability to find a difference. Therefore, we used nonparametric statistics robust to the variation in values higher than the median.

The K-9 resistance task group may have had a blunted response because the volunteers were not blinded to the fact that they were wearing a protective bite suit and only experienced pressure or pinching from the bite. Additionally, several of the study subjects complained when they were not assigned to the dog bite task (they were looking forward to trying that task). We believe that this may have also yielded an underestimation of the true effects that would occur in a real LEA K-9 custodial arrest interaction (since field situations would more likely involve fear).

The time limit chosen for the heavy bag resistance task group in the study was felt to be a conservative estimate of the time to physically restrain a combative subject. In situations in which the LEAs and suspects are not evenly matched, or in cases of supranormal ability to offer resistance (e.g., drug intoxication or excited delirium syndrome), it would likely take much longer than 45 seconds to physically restrain the subject. It appeared that the heavy bag task group was fatigued after 30 seconds. We may have underestimated the true physiologic burden of a real field encounter in the heavy bag group.

CONCLUSIONS

We believe that this is the first study to evaluate the human acid/base and catecholamine response of a resisting subject during a simulated law enforcement authority custodial arrest encounter. This physiology may be important in understanding the root cause of arrest-related death events. The actions of physically resisting and fleeing appear to be the most harmful with respect to acid/base and catecholamine physiology. This suggests that tactics and tools that limit this type of activity may be important in limiting the effect of the custodial arrest process on suspects.

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